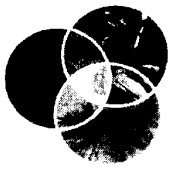
Overview



Cognitive aspects of depression

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Depression is a prevalent and impairing psychiatric disorder that affects how we feel and how we think about ourselves and the world around us. Cognitive theories of depression have long posited that various thought processes are involved in the development, maintenance, and recurrence of depressive episodes. Contemporary research has utilized experimental procedures to examine cognitive processes in depressed individuals as well as the nature of the relation of these processes to the emotion dysregulation that is central to the disorder. For example, investigators have assessed the ways in which depression alters aspects of information processing, including attention and perception, interpretation, and memory processes; this research has generated relatively consistent findings. In addition, researchers have attempted to identify and elucidate the cognitive mechanisms that may link these biases in information processing to emotion dysregulation in depression. These mechanisms include inhibitory processes and deficits in working memory, ruminative responses to negative mood states, and the inability to use positive and rewarding stimuli to regulate negative mood. Results of these investigations converge on the formulation that depression is associated with increased elaboration of negative information, difficulties in cognitive control when processing this information, and difficulties disengaging from this information. Research examining cognitive aspects of depression not only enhances our understanding of this common and costly disorder, but also has implications for the treatment of depression and for future investigations of the biological foundations of this disorder. © 2012 John Wiley & Sons, Ltd.

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INTRODUCTION

Major Depressive Disorder (MDD) is a debilitating psychiatric condition that affects almost 20% of the American population at some point in their lifetime.[1] In fact, the World Health Organization Global Burden of Disease Study ranked depression as the single most burdensome disease worldwide with respect to total disability-adjusted years among midlife adults.[2] MDD comprises a range of emotional, cognitive, and behavioral symptoms, including the core features of persistent depressed mood and decreased interest or pleasure in usually enjoyable activities. Other common symptoms of depression are psychomotor agitation or retardation, marked weight or appetite changes, insomnia or hypersomnia, fatigue, extreme feelings of guilt or worthlessness, concentration difficulties, and suicidal ideation. To meet *Diagnostic and Statistical Manual of Mental Disorders*[3] criteria for MDD, a subset of these symptoms must be present concurrently for at least a 2-week period, referred to as a Major Depressive Episode. Depression is a highly recurrent disorder; more than 75% of depressed individuals will relapse within 2 years of recovery from an episode.[4] This high rate of recurrence suggests that there are specific factors that increase the likelihood that individuals will experience repeated depressive episodes. In this review, we focus on cognitive aspects of depression as one class of such factors.

Cognitive theories of depression originated over 40 years ago and proceeded to stimulate multiple lines of research. Beck[5] posited that individuals who are vulnerable to depression have memory representations, or schemas, that lead them to view

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their environment in systematically negative ways. Beck postulated further that when these biases in cognitive processing interact with a negative life event, or stressor, these individuals are prone to a cycle of negative automatic thoughts about the self, the world, and the future (the 'cognitive triad'), and concomitant negative mood. Moreover, Beck and others developed interventions to change patterns of maladaptive thoughts and behaviors, and posited that these changes would lead to improvement in other symptoms of depression. Indeed, cognitive–behavioral therapy is one of the most effective treatments for MDD.[6]

Since Beck's initial formulation, researchers and clinicians alike have been examining cognition and emotion in MDD and have expanded and refined cognitive theories of depression.[7–10] Collectively, this work has documented that depressed and nondepressed individuals differ not only in the content of their thoughts, but also with respect to cognitive deficits and biases in the processing of information. Below, we briefly review findings regarding general cognitive deficits in depression. Next, we review studies of cognitive biases in depression, including biases in attention, interpretation, and memory. We then describe research that aims to identify the mechanisms linking cognitive difficulties and biases to emotion dysregulation in depression. Earlier reviews, particularly those of Williams et al.[10] and Mathews and MacLeod,[8] have nicely summarized many of these findings. In this chapter, we integrate more recent research and discuss the conclusions and limitations of work in this field to date. Building on previous reviews, we formulate a characterization of depression that underscores its several key cognitive features. Finally, we briefly discuss the implications of extant research both for the treatment of depression and for future integrative investigations of psychological and biological functioning in this disorder.

COGNITIVE DEFICITS IN DEPRESSION

Because diagnostic criteria for MDD include cognitive deficits, investigators have examined the nature of difficulties in cognitive functioning that are associated with depression. The resource allocation hypothesis postulates that depression uses cognitive resources and reduces cognitive capacity, thereby leading to difficulties engaging in effortful cognitive tasks.[11] Similarly, the affective interference hypothesis postulates that because depression involves a preoccupation with emotional material, performance will be selectively impaired on cognitive tasks that require individuals to ignore emotional information.[12] Supporting the affective interference hypothesis, Hertel[13] notes that depressed persons have been found to perform similarly to non-depressed individuals on structured cognitive tasks, but to exhibit greater attention and memory impairments on unconstrained tasks that provide more opportunities to engage in negative, task-irrelevant thoughts.

Additional empirical work has examined whether depression is associated with deficits in working memory (WM), the system involved in the active maintenance and manipulation of information.[14] Overall, findings of studies using a variety of tasks that assess the functioning of WM have been mixed. Consistent with the postulates of the affective interference hypothesis, several researchers have concluded that MDD is characterized by specific deficits in executive and attentional control, as reflected in a diminished ability to attend to task-relevant stimuli and inhibit task-irrelevant information. In contrast, individuals diagnosed with severe depression with psychotic features as well as elderly depressed individuals appear to be characterized by broader cognitive deficits.[15] We should note that in this area of research, it is critical that cognitive deficits are distinguished from a lack of task motivation or effort that is often experienced and exhibited by depressed individuals.[16]

Thus, although there is little evidence to support the position that depression is characterized by pervasive cognitive deficits, depressed individuals have been found to exhibit greater difficulties than do their nondepressed counterparts in unconstrained situations that require greater cognitive control or executive functioning, reflected in the ability to attend to task-relevant stimuli and inhibit irrelevant material in WM. Therefore, as we discuss in greater detail below, MDD appears to be characterized by impairments in cognitive control and inhibition.

COGNITIVE BIASES IN DEPRESSION

In addition to examining cognitive deficits, investigating how depression biases the processing of emotional information, particularly toward negative material, contributes to our understanding of cognition in depression. Recent cognitive models of depression posit that depression is associated with biases across several stages of information processing, including perception and attention, interpretation, and memory.[8] As we review below, however, findings from this research indicate that depression is associated consistently only with specific types of cognitive biases.

Biases in Perception and Attention

Automatic Biases

Biases in perception and in early stages of attention in depression have been assessed by using stimuli that are presented subliminally or very quickly, or by using stimuli that have low emotional intensity. Investigators have utilized stimuli relevant to depression as well as to anxiety, and have generally compared cognitive performance for responding to these emotional stimuli with performance for responding to neutral or other control stimuli. In general, this research has not consistently documented the operation of automatic biases in perception and attention in depression.[8]

For example, the Stroop task has been used to assess reaction time, or latency, to name a nonsemantic attribute of a word, such as the ink color in which it is presented. In the subliminal version of this task, words are masked so that participants are not consciously aware of the content of the words. In a typical paradigm used with depressed participants, a series of emotional words and neutral words are presented; researchers assume that if depressed individuals have an automatic bias toward emotional stimuli, they will be slower to name the ink colors of the emotional stimuli than will non-depressed individuals because of the greater attentional interference caused by the content of words for depressed individuals. In an early study, Mogg et al.[17] did not find depression-associated differences in the latency to name the colors of subliminally presented negative emotional words. More recently, Lim and Kim[18] also failed to find differences between depressed and non-depressed participants using subliminally presented anxiety-relevant (threat-related) words.

The dot probe, or visual probe, task involves the brief or masked presentation of a pair of stimuli (words or faces) on the left and right sides of a computer monitor. In studies of depression, one stimulus is neutral and the other stimulus is emotional. These stimuli are quickly followed by the presentation of a probe (e.g., a dot) on the same side of the screen as one of the stimuli. Across numerous trials, participants indicate the location (i.e., left or right) of the probe. Allocation of attention on each trial is inferred from response latency: a shorter response latency is assumed to indicate that attention was directed on that trial toward the stimulus (neutral or emotional) that had been presented on the same side as the probe. In a study using subliminally presented anxiety-relevant words, Mathews et al.[19] failed to find differences between depressed and non-depressed participants. Similarly, Mogg et al.[20] found evidence for a negative attentional bias in participants diagnosed with an anxiety disorder, but not in participants diagnosed with depression.

A small number of studies have used lexical decision tasks in which participants must indicate whether or not a stimulus is a word. In the subliminal version of the task, participants' decisions are preceded by the subliminal presentation of a prime emotional word or neutral word. Although findings using this task have been mixed, in one study that reported significant results, Bradley et al.[21] found that participants with higher levels of negative affect exhibited stronger priming effects for depression-relevant words, and this priming effect was more closely related to level of depression than to level of anxiety.

Two studies have used dichotic listening tasks to examine whether negative stimuli interfere with depressed individuals' attention to ongoing activities. Ingram et al.[22] reported that, following a negative mood induction, remitted depressed participants made more shadowing errors than did non-depressed participants when negative or positive stimuli were presented in the unattended auditory channel, thus indicating greater interference of these stimuli in ongoing processing of material in the attended channel. McCabe and Gotlib[23] integrated a dichotic listening task with another, secondary, task involving responding to a light probe, and found that depressed participants took longer to respond to the probe when negative words were presented in the unattended channel than when neutral and positive words were presented. This effect was not evident following recovery from the depressive episode, raising the issue of the state-dependent nature of this cognitive bias.

Because most of the relevant studies in this area have used words as experimental stimuli, it is not clear whether different results would be obtained for subliminally presented emotional faces or for other types of images. It will be important in future investigations to examine the automatic or early processing of facial expressions in depression, given that facial stimuli are salient social cues,[24] and that their correct identification aids in adaptive interpretation of situations and behavioral responses. In this regard, Joormann and Gotlib[25] found that when they were presented with facial stimuli showing subtle expressions of emotion, depressed participants exhibited greater difficulty than did non-depressed participants in identifying subtle positive emotional expressions. Clearly, further systematic examination of automatic biases in attention to emotional faces and other relevant stimuli in depressed individuals is warranted.

Elaborative Biases

Although depression does not appear to be characterized reliably by biases in early stages of attention, evidence of cognitive biases at later, more elaborative, stages of attention is more consistent. These studies have typically used the modified Stroop task or dot-probe task with stimuli presented for longer durations. For example, using the modified Stroop task, Gotlib and Cane[26] presented words at supraliminal durations of 1500 ms, long enough for participants to be aware of their content. The authors found that, prior to receiving treatment, depressed participants exhibited longer response latencies for negative words than did non-depressed participants. Other investigators, however, have failed to find this effect in depressed individuals.[17] Studies in which self-descriptive words were presented to participants have reported somewhat stronger findings. For instance, Segal and colleagues found that depressed participants showed increased interference for negative self-descriptive words following the presentation of negative self-descriptive phrase primes.[27] In their meta-analysis of studies of attentional biases in depression, Peckham et al.[28] reported only marginally significant differences between depressed and non-depressed participants on both subliminal and supraliminal versions of the modified Stroop task.

Peckham et al.[28] reported that group differences were more robust for the dot-probe task. For instance, Bradley et al.[29] studied participants with naturally occurring or induced dysphoria, a subclinical form of depression typically assessed via self-reported depressive symptomatology. These researchers found dysphoria-related biases toward negative words when stimuli were presented for 500 or 1000 ms, but not when they were presented subliminally. Donaldson et al.[30] replicated this finding in depressed participants using negative words presented for 1000 ms, but not for 500 ms. Other researchers have also failed to find group differences in dot-probe negative biases at 500 ms.[19] In a dot-probe study utilizing emotional faces as stimuli, Gotlib et al.[31] reported that depressed participants exhibited an attentional bias for sad faces presented for 1000 ms. This attentional bias was specific to sad faces, and was not found for angry or happy faces. Subsequent studies have extended these findings, reporting biases toward sad faces in remitted depressed participants[32] and in non-disordered girls at high risk for depression by virtue of their mothers' diagnosis of MDD.[33] We should note that not all studies have found significant depression-related effects for supraliminally presented stimuli on the dot-probe task.[20] Overall, however, studies using both verbal and facial stimuli in this task report evidence that depression is associated with biases toward negative emotional information at later, or more elaborative, stages of attentional processing. In this context, Koster et al.[34] recently utilized a spatial-cueing task and found that dysphoric participants showed an attentional bias for negative words under a condition that allowed for elaborative processing, but not under a condition in which elaborative processing was prevented. Moreover, studies reviewed above indicate that this elaborative attentional bias may not only characterize, or be a symptom of, depression, but may also represent a cognitive vulnerability for repeated depressive episodes.

Integrating the results of studies that have used subliminal and/or supraliminal stimulus presentations, the research to date suggests that depressed individuals are not characterized by biases in all aspects of perceptual or attentional processing. That is, depressed individuals do not consistently exhibit an automatic bias toward negative information, but once this information has captured their attention, they have difficulties disengaging from it. This proposition is consistent with other evidence indicating that, compared to their non-depressed peers, depressed participants exhibit longer durations of looking at negative emotional stimuli,[35] difficulties disengaging from negative images,[36] and increased task distraction due to negative stimuli.[37]

Interpretation Biases

In a small body of work, investigators have examined biases in the interpretation of ambiguous information in depression. In an early experiment, Butler and Mathews[38] presented participants with multiple ambiguous scenarios and found that depressed participants ranked negative interpretations of these scenarios higher than they did other possible interpretations. Since this study, other investigations have failed to demonstrate a depression-related interpretation bias even when measured following a negative mood induction.[39] In an innovative study, Lawson et al.[40] utilized a psychophysiological measure, startle magnitude, during imagery elicited by ambiguous text and were able to index more negative interpretations in depressed participants than in non-depressed participants. Reporting results suggesting that biases in interpretation serve as a cognitive vulnerability factor for depression, Rude et al.[41] found that greater interpretation bias predicted a subsequent increase in depressive symptoms in a sample of undergraduate students. Finally, Dearing and Gotlib[42] reported evidence of a negative interpretation bias in a sample of non-depressed girls at high risk for depression due to their

mothers' diagnosis of MDD. Thus the findings in this area are mixed, and further research is needed examining negative interpretation biases in depression.

Memory Biases

Of the three components of information processing reviewed, the strongest empirical support has been found for depression-related biases in memory processes.[43] For example, one of the most robust and consistent findings involves depressed participants' preferential recall of negative relative to positive information.[8] This extends to memory bias for sad faces in depression.[44] In contrast, non-depressed participants typically exhibit a bias for positive material. Findings are more reliable for studies measuring explicit memory in depression than for investigations of implicit memory. Furthermore, Watkins[45] argued that memory biases in depression are observed under conditions of semantic processing rather than of perceptual processing and, therefore, appear largely due to the increased elaboration of negative information that characterizes depressed individuals. In this context, Koster et al.[34] recently reported that in dysphoric participants, attentional bias toward negative words in a condition that allowed for elaborative processing predicted the number of negative words subsequently recalled.

In addition to a memory bias for negative material, Williams et al.[46] found that individuals diagnosed with depression recalled more generic or overgeneral memories than did their non-depressed counterparts, even when given instruction to recall specific details. Raes et al.[47] further demonstrated that overgeneral memory was correlated with cognitive deficits and longer depressive episode duration. Similarly, more overgeneral positive memories have been found to predict both poorer recovery from depression 7 months later[48] and a longer more delay in recovery from affective disorders.[49] Notably, this phenomenon has been shown to persist outside of depressive episodes and to predict future onset of episodes in women with postpartum depression.[50] Although not all studies have been able to document this effect, depression does appear to be characterized not only by an increased accessibility of negative material in memory, but also by the recall of overgeneral memories.

Importantly, the tendency to elaborate negative emotional information, memory bias for this information, and the recall of overgeneral memories may be associated with other cognitive aspects of depression. For instance, Williams[51] hypothesized that depressed individuals may utilize overgeneral memory to decrease the distress associated with negative memories. In a series of experiments, Dalgleish et al.[52] demonstrated that the decreased executive control associated with depression may underlie this effect. Similarly, Williams et al.[46] argued that the recall of overgeneral memories may have its underpinnings in difficulties in cognitive control or inhibition. Given that difficulties in cognitive control and inhibition have been documented in a number of areas of research in depression, we discuss this research further in Box 1.

BOX 1

COGNITIVE BIASES IN ANXIETY DISORDERS

Many of the paradigms used to study cognitive biases in depression have also been utilized in research on anxiety disorders. Anxiety disorders include Panic Disorder, Social Phobia, Specific Phobia, Generalized Anxiety Disorder (GAD), Posttraumatic Stress Disorder (PTSD), and Obsessive–Compulsive Disorder (OCD). Interestingly, although depression is frequently comorbid with anxiety disorders,[53] investigators have found key differences in their cognitive characteristics. Williams et al.[10] first distinguished between anxiety-related biases in attention and depression-related biases in elaboration and recall. Indeed, in contrast to depressive disorders, anxiety disorders have been shown to be characterized by cognitive biases toward threat-relevant information in the early, automatic stages of perception and of attention.[8] For example, on the subliminal Stroop task, individuals with various anxiety disorders exhibit longer response latencies for threat-relevant words than for neutral words.[17,20] Similarly, investigations using the subliminal dot-probe task have found that individuals with anxiety disorders attend automatically to threat-relevant material, even though its content is outside conscious awareness.[20] As we review in this article, depressed individuals do not appear to orient automatically toward negative information; instead, they exhibit biases in the subsequent elaboration of this material and difficulties disengaging from negative information. In addition, whereas depression has been found to be associated with memory biases and difficulties, individuals with anxiety disorders do not consistently exhibit memory biases for threat-relevant information.[54] Thus, although depression and anxiety disorders are each characterized by biases toward negative information, they differ in the precise nature of these cognitive biases.

INHIBITION AND COGNITIVE CONTROL IN DEPRESSION

Depression is associated with the increased elaboration of and difficulties disengaging from negative material. The ability to inhibit the processing of negative irrelevant information is central to humans' ability to respond adaptively to tasks and demands in their ever-changing environments. According to theoretical models of WM, our WM system has a limited capacity; therefore, at any point in time it reflects the focus of our attention and the representations that are currently and temporarily activated in our awareness.[55] Theorists have proposed that executive processes control and update the contents of WM,[56] serving to protect WM from intrusions of irrelevant information and discarding information that is no longer relevant, thereby resolving interference in WM. As we reviewed briefly above, depression appears to be characterized by specific difficulties in executive functioning and attentional control. The experience of intrusive thoughts in depression may further signal difficulties in interference resolution. Moreover, as we discuss in more detail below, deficits in cognitive control and inhibition may be associated with the elaborative and memory biases documented in depressed individuals and facilitate the repetitive negative thinking, or rumination, that has been found to characterize depressed individuals.[57]

As is the case with studies of cognitive biases in depression, experimental tasks assessing difficulties in cognitive control and inhibition in depression have often been adapted from paradigms developed in basic cognitive science. For example, the negative priming task was originally designed to isolate inhibition accounts of selective attention.[58] In this task, participants are presented simultaneously with a target (e.g., a word written in blue) and a distractor (e.g., a word written in red) and are instructed to respond to (e.g., name) the target. Negative priming occurs when, on the subsequent trial, the previous distractor becomes the new target. The latency or delay in responding to that target, which was the previous to-be-ignored distractor, relative to a novel target, indexes the strength of cognitive inhibition on the previous trial. In the first study to use this task with depressed individuals, Linville[59] found that, in contrast to non-depressed participants who were slower in responding to targets that they were previously instructed to ignore, depressed participants did not exhibit this effect. Given that inhibitory deficits in depression may be even more pronounced for emotional information, investigators have recently developed and used a negative affective priming task. In this version of the task, stimuli are emotional, such as negative and positive words, and are used as both targets and distractors. In an initial study, Joormann[60] demonstrated that participants with dysphoria and a history of depression showed impaired inhibition of negative adjectives. That is, these participants responded more quickly to negatively valenced targets that followed negative distractors on preceding trials. Moreover, this reduced inhibitory ability in dysphoria was specific to negative words. In subsequent studies, participants with high self-reported rumination exhibited this effect even after controlling for level of depressive symptoms.[61] Similar results have been reported in studies using emotional faces as stimuli; Goeleven et al.[62] found that, compared with non-depressed participants, depressed individuals exhibited decreased inhibition for sad but not for happy facial expressions. Extrapolating from these findings to theoretical accounts of WM, results of both the original and the affective versions of the negative priming task suggest that depressed individuals have difficulty keeping irrelevant material, and negative material in particular, from entering WM.

Another critical aspect of cognitive inhibition involves the ability to discard previously relevant material from WM. Depression-related difficulties in this form of cognitive processing may help to explain why, following negative events or mood states, depressed individuals are prone to experience recurrent, persistent, uncontrollable negative thoughts. Joormann and Gotlib[63] recently used a modified Sternberg task to examine the ability of depressed individuals to expel valenced material from WM. In this task, participants are presented with two simultaneous lists of emotional words and are instructed to memorize the lists. Next, a cue signals to participants the list that is relevant for an upcoming recognition memory task. On each trial of the recognition test, participants indicate whether a probe word came from the relevant list; in order to do this, participants must correctly reject words from the no-longer-relevant list. The ability to remove previously relevant information from WM is operationalized as the difference in average reaction times between probes from the previously relevant list and never-before-seen probes.[64] Joormann and Gotlib[63] found that, relative to non-depressed participants, participants with MDD exhibited a longer latency for negative probes from previously relevant lists than for novel negative probes. This effect was not found for positive probes. Similar to findings for the negative affective priming task, this effect was associated with self-reported rumination, even after controlling for level of depressive symptoms. Joormann et al.[65] designed an experiment to examine

both access of information to WM and discarding irrelevant information from WM. Depressed and non-depressed participants completed two types of tasks: an 'ignore' task in which they were instructed to memorize a series of emotional words and to ignore other concurrently presented words, and a 'suppress' task in which they were told to forget a portion of the previously memorized words. Following each type of task, participants were administered an interference resolution test in which they were presented with a word and asked to indicate whether it was from the target set (i.e., a word that they had been instructed to remember). Joormann et al. found that depressed participants exhibited longer latencies than did non-depressed participants to negative words that they had been instructed to suppress, but not to words that they had been instructed to ignore. These results suggest that depressed individuals have greater difficulties than do their non-depressed peers only in removing negative material from WM. Again, these difficulties were associated with self-reported rumination.

A growing body of evidence suggests that these difficulties in cognitive control that are associated with depression extend to impairments in the intentional forgetting of information. This postulate has been examined in studies using the directed-forgetting task, in which participants are first instructed to study particular material and are subsequently told to forget that information.[66] Cottencin et al.[67] used neutral material and documented that depressed participants exhibited increased recall of to-be-forgotten words and decreased recall of to-be-remembered words, suggestive of a more general impairment in cognitive control. However, in a study utilizing negative and positive adjectives, Power et al.[68] demonstrated greater facilitation, or less successful inhibition, for to-be-forgotten negative words only when they were processed by depressed participants in a self-referential manner. Joormann and Tran[69] found that participants high in self-reported rumination similarly showed decreased forgetting of negative words. These participants also exhibited increased incorrect recall of negative words that had not been previously presented, a finding that was replicated in a sample of depressed participants.[70]

Finally, research has been conducted examining the performance of depressed individuals on an intentional forgetting task adapted from Anderson and Green.[71] Hertel and Gerstle[72] conducted an experiment in which participants first learned a series of word pairs consisting of one neutral noun (target) and one positive or negative adjective (cue). In this manner, the cues served to imbue the targets with emotional valence. On the subsequent trials, participants were presented with successive cues and practice recalling or suppressing (i.e., not thinking about) their associated targets. The final test phase assessed participants' recall for the targets. The authors evidenced greater recall for to-be-suppressed words in dysphoric than in non-dysphoric participants, with a particular trend for to-be-suppressed negative words. Furthermore, the extent of forgetting was associated with self-reported rumination, such that a greater number of to-be-suppressed words were recalled by participants who reported more rumination when sad. Two additional studies[73,74] used a similar design but presented neutral cues paired with emotional targets and found that although these impairments in the inhibition of negative material appear to extend to depressed individuals, depressed participants can be trained to improve their forgetting of negative material when instructed to use specific strategies (i.e., thought substitutes).

In summary, the results of research on cognitive inhibition are generally consistent with the findings we reviewed earlier for cognitive biases. Rather than suggesting a generalized bias at all stages of information processing or a global deficit in cognitive processing, the results of these studies reveal that depressed individuals are particularly vulnerable to elaborating on negative material in their environment, and that they experience difficulty disengaging from and inhibiting their processing of this material. As evidenced in multiple studies, these impairments are significantly associated with rumination, a specific style of thinking that involves the recurrence or recycling of thoughts in response to negative affect and that has been implicated as a vulnerability factor for depression.[57] Below, we review research that has attempted to link cognitive difficulties and biases to the dysregulation of emotion that has been found to characterize depressed persons.

COGNITION AND EMOTION REGULATION IN DEPRESSION

Multiple factors are likely involved in the persistent negative affect experienced by depressed individuals. Cognitive models of emotion underscore the role of cognitive appraisals in individuals' experience and regulation of emotions. With respect to depression, cognitive deficits and biases in the processing of emotional information may impair depressed individuals' ability to adaptively regulate their emotions.[75] In particular, difficulties in controlling the negative, mood-congruent contents of WM may impair the ability of depressed persons to flexibly reappraise or reinterpret life events.[76] For example, deficits in

removing negative no-longer-relevant material from WM may lead to difficulties in processing new information. In addition, attention and memory biases toward negative information may engender a rigid pattern of negative appraisals of situations that make it more difficult to reappraise events or regulate emotions.[77] The repeated operation of cognitive biases may lead to persistent maladaptive emotion regulation strategies. Thus, although the various cognitive processes implicated in depression are not posited to affect emotion regulation directly, they may have important consequences for the effectiveness of the regulation of emotion. This formulation is supported by findings that both currently depressed and remitted depressed participants exhibit impaired emotion regulation abilities.[78]

Several studies have investigated in a more causal manner the postulate that difficulties disengaging from negative information can increase emotion reactivity and decrease adaptive emotion regulation. For instance, Ellenbogen et al.[79] found that decreased ability to disengage attention from supraliminal dysphoric images was associated with increased negative mood reactivity to a subsequent stressor, although it did not affect neuroendocrine reactivity. Although the results of this work are promising, it is important to conduct studies examining this postulate in samples of depressed individuals.

Memory biases may also influence emotion regulation in significant ways—the accessibility of negative material in memory may influence emotional responses and emotion regulation strategies. In fact, emotional memories themselves may serve to regulate affect. For example, investigators have shown that memory for unpleasant events diminishes more quickly than does memory for pleasant events and, furthermore, that this disproportionate fading is positively related to happiness.[80] Recalling positive memories of life events, or mood-incongruent recall, can repair or improve induced depressed mood.[81] Joormann et al.[82] found that depressed individuals are less able than are their non-depressed peers to use positive autobiographical memories to regulate induced negative mood.

Finally, as suggested by multiple lines of research reviewed above, inhibitory deficits may be central to the occurrence of rumination in depressed individuals. In a systematic program of research, Nolen-Hoeksema and colleagues have demonstrated that rumination not only characterizes depression, but also predicts more severe depressive symptoms and the later onset of depressive episodes. Furthermore, this research group has shown that a greater tendency toward rumination, when interacting with negative cognitive biases or styles, predicts a longer duration of depressive episodes.[57] In support of a relation between rumination and cognitive control, Davis and Nolen-Hoeksema[83] found that ruminators made significantly more errors on a task assessing executive control and cognitive flexibility (the Wisconsin Card Sorting Task). Similarly, Joormann et al.[84] found that depressed participants exhibited more difficulties than did controls in manipulating words, particularly negative words, in WM. Moreover, within the depressed group, greater self-reported rumination predicted greater difficulties manipulating negative words, suggesting that rumination is associated with impaired cognitive control in depression. In additional examples reviewed above, investigators have found significant correlations between self-reported rumination and difficulties in cognitive inhibition.[61,63] Other evidence suggests that the process of rumination exacerbates cognitive biases in depression. In two studies, dysphoric participants who were induced to ruminate exhibited poorer problem-solving strategies, increased negative interpretation biases,[85] and increased recall of negative autobiographical memories,[86] relative to dysphoric participants who were not induced to ruminate. These processes likely affect individuals' emotional experience; in a recent experiment with dysphoric participants, Williams and Moulds[87] found that induced rumination, relative to induced distraction, led participants to rate their intrusive memories as more distressing and to report experiencing more negative mood. Thus, across multiple studies, cognitive aspects of depression appear to be tightly linked to depressive rumination. Clearly, given the vicious cycles of thought and feeling that may be engendered by the interaction of cognitive deficits, cognitive biases, and emotion dysregulation in depression, this is an important area for future research.

IMPLICATIONS FOR THE TREATMENT OF DEPRESSION

The cognitive biases and deficits that have been found to characterize depression represent important targets for intervention. It follows from the models and the research we reviewed above that ameliorating dysfunction at the cognitive level may help to reduce depressive symptomatology and other features of depression. In this regard, it is important to note that investigators examining anxiety disorders have recently tailored the typical dot-probe paradigm to train participants to disengage from negative anxiety-relevant stimuli.[88] In a common version of this training paradigm, a pair of stimuli is presented to the participant, but the subsequent probe is presented more

frequently in the location of the neutral stimulus than of the negative stimulus, thereby directing participants' attention away from the negative stimulus. This training task has been found to reduce subsequent negative affect reactivity to a laboratory stressor.[89] In two recent experiments with dysphoric and depressed samples, Baert et al.[90] compared this type of attention training away from negative stimuli to a condition involving no contingency between the stimulus valence and probe location. The authors found that, following the training, participants with lower initial levels of severity of depressive symptoms exhibited greater improvements in symptomatology, whereas participants with higher initial levels of symptom severity did not exhibit such improvement. It also appears that positive attentional biases can be trained and can decrease later attention to negative images.[91] Given these promising findings, therefore, further research is warranted examining different types of attention training interventions with individuals at varying levels of depression severity.

Cognitive biases may also be trained at the stage of interpretation. In a recent study, Holmes et al.[92] found that training positive interpretation biases utilizing imagery served to reduce the adverse response of non-clinical participants to induced negative mood. Similarly, Tran et al.[93] found that training a positive interpretation bias led participants to recall more positive than negative intrusions, i.e., information that was not presented at earlier encoding. Importantly, however, no studies to date have examined the effects of training more positive attention or interpretation in depressed individuals; this is a critical next step for research in this area.

With respect to overgeneral memory, Watkins et al.[94] demonstrated that training dysphoric participants in more concrete and less overgeneral memory led to significant decreases in both depressive symptomatology and frequency of rumination. In addition, Raes et al.[95] reported initial evidence that training depressed participants to recall more specific memories generated improvements in their memory retrieval, level of rumination, and quality of problem solving. Finally, Siegle et al.[96] reported preliminary findings for an intervention aimed at improving cognitive control in severely depressed individuals. This intervention led to similar decreases in both depressive symptomatology and rumination. Therefore, cognitive control and overgeneral memory may similarly be amenable to modification. Taken together, these findings underscore the role of difficulties in cognitive control in emotion dysregulation in depression, and highlight ways to target these mechanisms using cognitive interventions.

CONCLUSION

As we have documented in this review, it is evident that depression changes the ways in which we think about ourselves and the world around us, and that the cognitive biases and deficits that reliably characterize depression influence maladaptive patterns of emotion dysregulation in this disorder. Assimilating the research to date, findings converge on the formulation that depression is characterized by the increased elaboration of negative material, difficulties disengaging from this material, and deficits in cognitive control when processing this material. Moreover, there is growing evidence that these cognitive factors may confer vulnerability for the development and recurrence of depressive episodes. In particular, the ability to control the contents of WM may have profound effects on people's ability to recover from negative events and from the experience of negative affect; moreover, difficulties in this domain of cognitive functioning may be related to the frequently observed problematic cycles of uncontrollable rumination and worsening negative mood in depression. A goal of future research is to map more explicitly and systematically these links between cognitive and affective aspects of depression.

In addition, future studies will benefit from the integration of biological aspects of depression associated with various observable components of the disorder. A growing body of empirical work has been conducted investigating the brain structures implicated in depression.[97] These studies have highlighted the roles of the limbic system (the amygdala, hippocampus, and parts of the anterior cingulate cortex) in depression, as well as the role of the dorsolateral prefrontal cortex in the regulation of emotion. As recently reviewed by Disner et al.,[98] depression appears to be characterized by hyperactivity of the limbic system along with decreased activity in cognitive control regions, consistent with the cognitive findings reviewed in this chapter, and with Beck's original formulation of cognition in depression. For example, in an important study, Johnstone et al.[99] demonstrated that during reappraisal of emotional images, nondepressed participants exhibited increased activation in the dorsolateral prefrontal cortex and decreased activation in the amygdala, whereas depressed participants did not. Thus, neural assessments may provide insight into the underpinnings of impairments in cognitive control and emotion regulation in depression. It is important to note, however, that the majority of studies examining neural aspects of depression are correlational in nature; research assessing high-risk populations will help to elucidate neural mechanisms

underlying vulnerability to depression.[100] In addition, genetic factors, in particular the short allele of a functioning 5′ promoter polymorphism of the serotonin transporter gene (5-HTTLPR), have been associated with the onset of depression following stressful life events[101]; indeed, there appear be similar associations between 5-HTTLPR and cognitive biases in depression.[102] Future integrative research in cognitive science must attempt to bridge genetic, neural, cognitive, and affective aspects of depression.

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